EXHIBIT A

DECLARATION OF FRANK L MARGOLIS, PH.D.

1. My name is Frank L. Margolis, Ph.D. I am over 18 years of age, of sound mind, and fully competent to make this declaration. I have personal knowledge of the facts stated herein and if called upon to do so, I could competently testify thereto.

2. I received my Ph.D. in Biochemistry from Columbia University, New York City, New York. I am a full professor at the University of Maryland School of Medicine where my primary appointment is in Anatomy and Neurobiology. I am also Director of the MS Program in Molecular Medicine.

3. My laboratory research involves the investigation of the vertebrate olfactory system. I have performed research into the biochemistry of the olfactory system for over thirty years. Describing part of that research, in 1981 I published *Neurotransmitter Biochemistry of the Mammalian Olfactory Bulb* in Biochemistry of Taste and Olfaction (Academic Press). In 1985 I published "*Carnosine Synthesis in Olfactory Tissue During Ontogeny: Effect of Exogenous β-Alanine*" in the Journal of Neurochemistry with a number of my colleagues.

4. In the 1981 publication, we did not measure muscle carnosine or beta-alanine in muscle tissue. In the 1985 publication, we describe an experiment where we injected beta-alanine into the intraperitoneal cavity, *i.e.*, through the peritoneal membrane into the abdomen, of a rodent. The beta-alanine was injected twice a day for 2-5 days at 22 mmol/kg bodyweight. This corresponds to about 1.96 grams of beta-alanine per kg body weight for each injection.

5. The beta-alanine was obtained from commercial sources. My understanding is that commercial beta-alanine is made chemically from acrylonitrile and ammonia. While beta-alanine in some mammals is made in the liver, commercial sources make it chemically. The MSDS for this non-natural beta-alanine indicates the oral toxicity (LD50) is 1000 mg/kg for the rat, or 1 gram/kg body weight. LD50 is an abbreviation for "Lethal Dose, 50%" or median lethal

dose, which is the amount of the substance required (usually per body weight) to kill 50% of the test population.

6. Beta-alanine is rapidly broken down in the body and excreted. In my studies, I injected the animals with aminooxyacetic acid about 15 minutes before injecting them with beta-alanine. I did this because aminooxyacetic acid inhibits the metabolic breakdown of beta-alanine, thereby keeping the level of beta-alanine high in the organism. Moreover, aminooxyacetic acid is a known anticonvulsive (see, *e.g.*, Exhibit 1).

7. I have reviewed U.S. Pat. Nos. 8,067,381 and 8,129,422, which I understand are the patents in suit. I have also reviewed what the accused infringer has said regarding my 1981 and 1985 publications. I do not believe my 1981 or 1985 publications provide any information that is relevant to the inventions claimed in the patents in suit for the reasons set forth below.

8. In my experiments, the mice were injected with beta-alanine intraperitoneally. Administration by injection is very different compared to oral administration. For example, uptake and bioavailability following injection will be rapid and almost 100%, following oral administration it will be a slower process and not all of the beta-alanine will be available as some will have been processed for excretion. Indeed, the metabolism of beta-alanine injected intraperitoneally is so rapid that in our 1985 research we had to inject the animals two times per day, each time at approximately two times the reported LD50 for rodents and in combination with aminooxyacetic acid to inhibit beta alanine degradation.

9. The doses in my 1985 research were at such high amounts, the rodents would not be expected to have responded in the same way they would respond to a lower physiologically safe dose. Administering comparably high levels in humans would be unacceptable, if not potentially lethal. Paresthesia, an unpleasant tingling and burning sensation is experienced by

many humans taking beta-alanine at doses as low as 20mg/kg, which is almost 100 times lower than the doses used in my study.

10. My 1985 study was primarily concerned with carnosine synthesis in the olfactory tissue. There is far less olfactory tissue than muscle tissue in rodents. One of our findings was that carnosine concentration in olfactory tissue is initially higher than carnosine concentration in muscle and that there is faster synthesis of carnosine in olfactory tissue because there is a higher concentration of the enzyme carnosine synthetase in olfactory tissue. Given this higher initial concentration and faster synthesis, it is likely that any beta-alanine will be used by this olfactory tissue first before other tissues, such as muscle. The reason carnosine is seen to rise in the muscle tissue in my 1985 study is due to the massive doses of beta-alanine that were given in conjunction with the aminooxyacetic acid.

11. It is my understanding that researchers in the field of exercise physiology believe that it is necessary for humans to consume physiologically safe and sufficient amounts of beta-alanine for at least 2-4 weeks to see any measurable effect on muscle tissue performance. My study injected rodents with beta-alanine for 2-5 days at toxicologically high levels. This is not a good model for extrapolation to humans because of the evolutionary metabolic and dietary differences. In my scientific opinion, my research would not have motivated individuals to discover the inventions described in the '381 and '422 patents to increase muscle performance in humans, or provided any material information relevant to those inventions.

12. To the extent that the Court has any questions regarding this declaration, I would be happy to make myself available by telephone or in person at the Court's convenience.

I declare under penalty of perjury under the laws of the United States that the foregoing is true and correct.

Signed: _____

Date: 7/4/13